Intracranial dermoid cysts: variations of radiological and clinical features

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Abstract: BACKGROUND: Intracranial dermoid cysts are uncommon, and their clinical features as well as surgical management differ from patient to patient. Dermoids are generally benign lesions, but may cause spontaneous complications such as meningitis and/or hydrocephalus due to rupture and epileptic seizures depending on their location. Little has been reported about characteristic imaging findings with resulting therapeutic considerations, and only a few reports exist about associated hydrocephalus. Imaging modalities have changed and can facilitate differential diagnosis and follow-up if applied correctly. In this paper, we attempt to contribute our clinical experience with the management of dermoid cysts. PATIENTS AND METHODS: The charts of five men and two women with intracranial dermoid cysts were retrospectively reviewed. The patients were treated between September 1993 and September 2006. Selected patients are presented in detail. RESULTS: Tumour location, size and radiographic characteristics varied in each patient. Clinical presentations comprised focal neurological deficits as well as epileptic seizures, persistent headache, mental changes and psycho-organic syndromes. One patient underwent delayed ventriculo-peritoneal shunting after ruptured fatty particles caused obstructive hydrocephalus. Despite dermoid rupture into the subarachnoid space, three patients never developed hydrocephalus. Diffuse vascular supra-tentorial lesions were seen in one patient as a result of aseptic meningitis. Diffusion-weighted imaging (DWI) hyperintensity in dermoids is related to decrease of water proton diffusion and should be used for both the diagnosis and follow-up of this lesion. CONCLUSION: Although dermoid cysts are known to be benign entities per se, their rupture can cause a wide range of symptoms including aseptic meningitis and/or hydrocephalus. This may be due to intraventricular obstruction and/or paraventricular compression. While rupture does not necessarily bring about hydrocephalus, radical removal of the tumour and close monitoring of ventricular size is required. Although not widely recognised as such, DWI is considered to be a useful imaging modality in the diagnosis and follow-up of dermoids.

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CLINICAL ARTICLE

Intracranial dermoid cysts: variations of radiological and clinical features

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Keywords Dermoid · Hydrocephalus · Olfactory disturbance · CT · MRI

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Introduction

Intracranial dermoids are rare congenital conditions that account for 0.04–0.6% of all intracranial tumours [18, 33]. Derived from ectopic cell remnants that are incorporated into the neural tube, they are usually located along the midline. Various clinical aspects such as presentation with epileptic seizures, meningitis, focal neurological deficits, hydrocephalus, sudden death and incidental diagnosis have been reported. These acute symptoms associated with the presence of dermoids, their localisation and their rupture are well documented [1, 2, 5, 14, 15, 17, 18, 20, 23, 26, 33, 40–42]. Diagnostic findings were evaluated in the past but

Table 1 Clinical details of patients with intracranial dermoids

Patient number	Age, sex	Presentation	Location	CT	MRI	Rupture	HCP	Therapy	Morbidity	Follow-up	Recurrence
1	19, m	Recurrent seizures, headache for 10 years, hyposmia	Left fronto-basal, intraventricular	Hypodense, some solid structures	N/a	Yes	No	Resection after 10 years	Hyposmia (preexisting)	Seizure 9 years after surgery	No
2	33, m	Seizure	Intrasellar, roof of orbit, right petrous bone, intraventricular	Hypodense with calcification	T1-hyperintensity (nonhomogenous), T2-hyperintensity, fatty particles T1-hyperintense	Yes	Yes	Subtotal resection, ventriculo-peritoneal shunt	No	18 months	No
3	16, f	Persistent posttraumatic headache	Parasellar, right suprasellar, intraventricular, basal cisterns	Hypodense	T1-hypointensity (nonhomogenous), T2-hyperintensity, Gd enhancement, fatty particles T1-hyperintense	Yes	No	Subtotal resection	Temporarily impaired vision	24 months	No
4	60, m	Headache, dizziness, ataxia, n/v, dysarthria, right facial paresis, dysmetria	Right posterior fossa with compression of 4th ventricle	Hypodense with calcification, leucencephalopathy	T1-hypointensity (nonhomogenous), T2-hyperintensity, Gd enhancement, DWI hyperintensity, leptomeningeal Gd enhancement, (convexity, Sylvian fissure, perimedullary)	No	No	Total resection	No	3 months	No
5	56, m	Acute paranoid-psychotic episode, anterograde amnesia, neuropsychological deficits	Suprasellar with compression of optic chiasm	Hypodense	T1-hypointensity, T2-hyperintensity, Gd enhancement, DWI hyperintensity, (inhomogeneous)	Yes	No	Total resection	No	6 months	No
6	19, f	Headache, complex partial epilepsy, blurred vision, n/v, dizzyness	Meckel's cave, Foramen ovale, (extradural)	Hypodense	T1-hyperintensity, mild enhancement, T2-hyperintensity, fat suppression, DWI hyperintensity, extradural	No	No	Total resection	No	24 months	No
7	52, m	25-year history of epilepsy (generalised), dizziness, diplopia, EEG with pathological spikes right temporal	Right temporo-mesial	N/a	T1-hypointensity (nonhomogenous), T2-hyperintensity, no Gd enhancement	No	No	Total resection	No	6 months	No

Gd Gadolineum, *N/a* not available, *HCP* hydrocephalus, *n/v* nausea/vomiting, *DWI* diffusion weighted imaging

show strong individual variation [4, 8, 9, 13, 22, 24, 27, 31, 32, 36, 38, 44, 45]. The aim of this report is to contribute to the understanding of this complex disease by presenting a series of patients with distinct characteristics and to provide a brief literature overview.

Patients and methods

We retrospectively reviewed 7 patients with intracranial dermoids who were treated in the Department of Neurosurgery at the University Hospital in Zurich, Switzerland, between 1993 and 2006. We excluded patients with the diagnosis of epidermoid or teratoma. Patients were analysed considering clinical profiles, radiological studies, intra-operative findings and development of hydrocephalus.

Results

The patients predominantly presented with headache and/or seizures (Table 1). Their ages ranged from 16–60 years and the median age was 33 years. There were 5 males and 2 females. All diagnoses of dermoid were verified by microscopy. Four of our patients had a history of epilepsy, and subsequent investigations revealed an intracranial tumour. Co-existing symptoms were tumour location-dependent and included hyposmia, cerebellar symptoms, blurred vision and dizziness. Three patients who did not present with signs of epilepsy suffered from headache and one patient had acute paranoid-psychotic episodes. One patient was operated on for a large frontal dermoid 10 years after initial presentation with epileptic seizures and headache. Right-sided anosmia was the leading pre-operative symptom in this patient. Radiological investigations showed intracranial masses at different locations. As the period of inclusion of patients is rather long (1993–2006), imaging protocols were not identical. Cranial computed tomography (CT) (Fig. 1) and magnetic resonance imaging (MRI) was obtained in all patients. Formerly, pre- and post-operative MRI contained standard protocols including T2-WI (Fig. 2), T1-WI and post-contrast-enhanced T1-WI. Meanwhile, these protocols were extended to also include DWI as these sequences were found useful in characterising these lesions. On MRI, the tumours typically presented with inhomogeneous T1-hypointensity, T2-hyperintensity and moderate contrast enhancement (Figs. 3, 4, 5 and 6). DWI may demonstrate a hyperintense lesion (Fig. 7). Due to tumour rupture, fatty components were also observed intraventricularly (lateral ventricles) or in the subarachnoid space in four patients. In patient 4, leptomeningeal enhancement as a sign of aseptic meningitis was detected

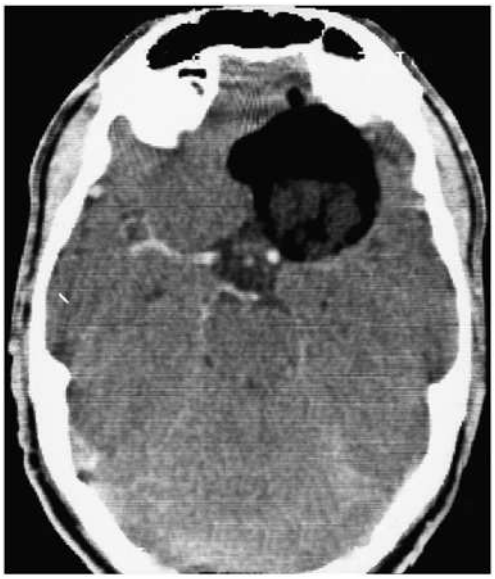

Fig. 1 Axial CT with left fronto-basal hypodense tumour adherent to the middle and anterior cerebral arteries (Patient 1)

in the basal cisterns, both Sylvian fissures, perimedullary and at the convexity. Multiple supratentorial vascular atherosclerotic lesions were visible.

Gross total resection could be achieved in five of the patients. In two patients, subtotal removal was performed in order to reduce peri-operative neurological morbidity. In patient 2, the solid tumour components around the sella were subtotally resected, and parts of the calcified

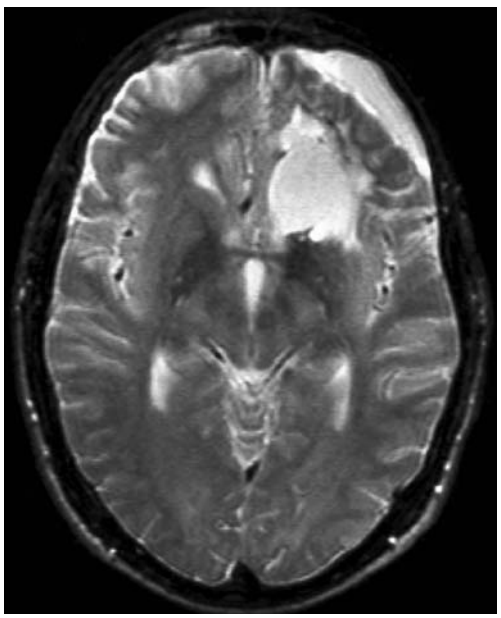

Fig. 2 One-year follow-up MRI (T2-WI) of Patient 1 with CSF-filled resection site and left subdural hygroma; no tumour rest or spilled components

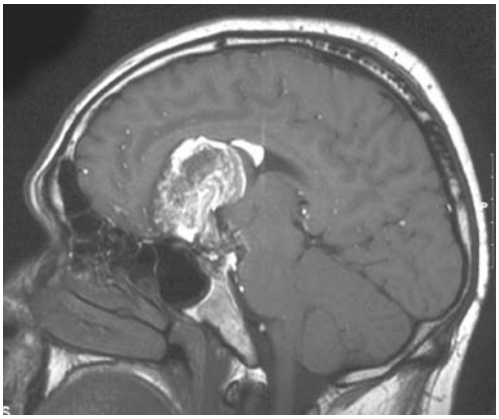

Fig. 3 Sagittal MRI (T1-WI) of a suprasellar dermoid with fronto-temporal extension and subarachnoid spreading of fatty particles (Patient 2)

tumour adherent to the internal carotid artery (ICA) and middle cerebral artery (MCA) were left *in situ*. Due to pre-operative rupture of the dermoid cyst, fatty particles obstructed intraventricular pathways at the level of the Foraminae of Monro and subsequently led to post-operative hydrocephalus which was treated with a ventriculo-peritoneal (VP) shunt. The intraventricular fatty particles (Fig. 4) in patient 3 were not removed. The patient never developed hydrocephalus and subtotal removal of the solid tumour components was accomplished. The patient has been clinically followed regarding subsequent obstruction of CSF pathways and development of hydrocephalus but is free of specific symptoms up to now. Within the tumour, dermoid-specific findings such as hair, sebaceous glands and cholesterol crystals were present in all cases and confirmed by histopathological examination. The radical resection of the dermoid included the capsule of the tumour.

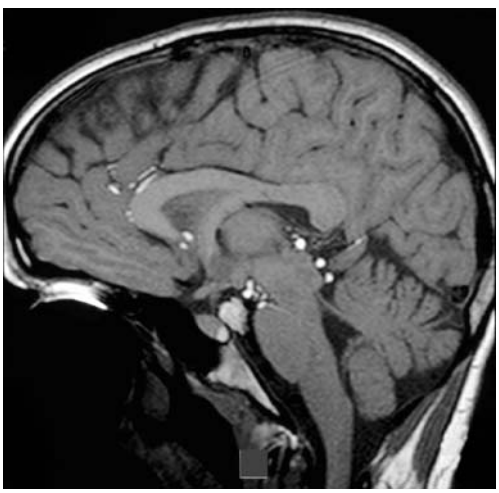

Fig. 4 Sagittal MRI (T1-WI) of an intra-, supra- and parasellar dermoid (Patient 2); spilling of fatty particles into the basal cisterns, ambient cistern, intraventricular and subarachnoid space with adhesion to the basilar artery

Fig. 5 Axial MRI (T2-WI) with hyperintensity of infratentorial dermoid with severe mass effect and compression of the 4th ventricle (Patient 4)

Post-operative morbidity was low. In patient 1 the pre-operative hyposmia did not improve and patient 3 temporarily experienced right-sided blurred vision which recovered fully to the pre-operative level. Due to pre-operative dermoid rupture, patient 2 developed an obstructive hydrocephalus that required a VP shunt.

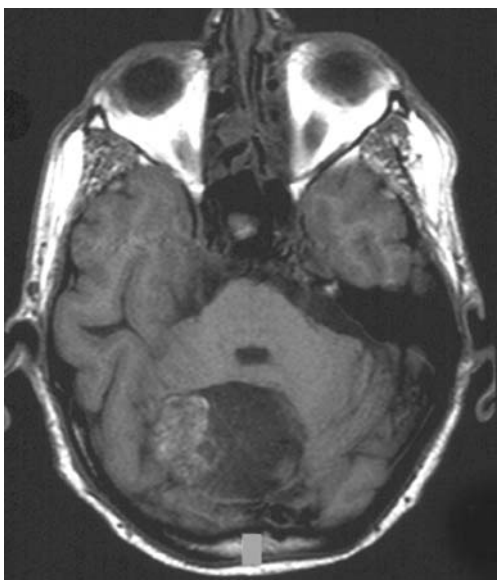

Fig. 6 Axial MRI (T1-WI) with mainly hypointense and laterally non-homogeneously iso- to hyperintense signal of dermoid (Patient 4)

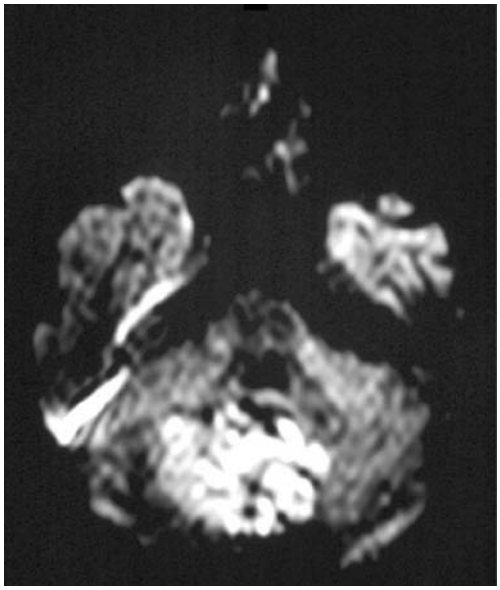

Fig. 7 Axial MRI (DWI) hyperintensity of some tumour areas, indicating decreased water proton diffusion as seen in keratin-rich lesions (Patient 4)

Follow-up periods varied from 3 months to 9 years after surgery. Clinically, all patients remained neurologically intact without any new neurological deficits or residual epilepsy that required medical treatment.

Discussion

Intracranial dermoids are reported to account for 0.04% to 0.6% [28, 33] of all intracranial tumours. As congenital dermoid and epidermoid cysts are formed by inclusion of ectodermal elements during neural tube closure, they may be diagnosed at a young age. Dermoids are composed of a thick fibrous capsule with a lining of stratified squamous epithelium, enclosing a thick viscous greenish-brown fluid. This fluid contains lipid metabolites, cholesterol crystals, whorls of hair, calcified areas and decomposed epithelial cells. Sebaceous or sweat glands may also be present [36]. Mesodermal origin is sometimes ascribed to dermoids, but this is a misconception, as dermoids derive from ectodermal cell lineages only. Other than true neoplasms, which grow by progressive cell division, they enlarge by desquamation of normal cells and secretion of dermal elements into a cystic cavity well circumscribed by a thick wall of connective tissue. Mainly capsular calcification is common, representing dystrophic changes or dental enamel, another ectodermal derivate. In contrast to epidermoids, dermoids are mostly located in or close to the midline, which is due to their earlier formation. A dermal sinus may be present with spinal, anterior and posterior fossa lesions [6].

Ruptured dermoids present with various manifestations. Patients with dermoid cysts may either remain asymptomatic or present with headaches, epileptic seizures, signs of compression of neighbouring tissues, aseptic meningitis, transient cerebral ischemia as a result of vasospasm and even olfactory halucinations [11, 14, 18–20, 25, 33, 40, 45–47]. After rupture of these cysts, their fatty content disperses into the subarachnoid space and may be present in any of the cisterns and ventricles [17, 23, 30, 36, 38, 39, 43]. Most often the rupture occurs spontaneously although trauma, such as a closed head injury, has been implicated in certain instances (see patient 2) [25]. In patients with generalised involvement of the meninges and/or subarachnoid distribution of dermoid particles, reactive vasospasm may occur. Vasospasm can also be a reaction to direct mechanical compression by the space-occupying lesions [1, 11, 14]. Rarely, intraventricular fat leads to rapidly developing occlusive hydrocephalus [17, 23, 24].

Certain radiographic characteristics have been described in the literature [22, 24, 36, 38, 45]. Osborn et al. concluded that dermoid contents usually resemble fat on CT and MRI whereas epidermoids resemble CSF [29]. The literature refers to dermoids mostly associated with T1-WI hyperintensity and T2-WI hypointensity due to a higher density of cholesterol within the tumour. Less commonly, dermoids may display signal characteristics like epidermoids, if the fatty contents are less dense. A CSF-like signal with T2-WI hyperintensity and T1-WI hypointensity may be present. Chen et al. described epidermoids as tumours with a strong DWI hyperintensity as was seen in patient 4 due to an effect of the keratinoid contents [7]. It was recommended to use fluid attenuated inversion recovery (FLAIR) sequences and DWI to distinguish epidermoids from cystic lesions, but as demonstrated by our patient 4, histopathologically confirmed dermoids can resemble epidermoids due to their bright signal on DWI [7, 10]. Only few studies on the DWI appearance of intracranial dermoids exist [3, 35]. Aksoy et al. reported a hyperintense DWI signal in a posterior fossa dermoid that pre-operatively mimicked an epidermoid. The solid components of the dermoid led to decreased diffusion within the tumour compared to purely cystic lesions [3]. Other authors also reported a few examples in which the CT appearance was hyperdense, and even partial rim enhancement has been documented [8, 9, 13, 21, 22, 27, 33]. Haemorrhage-like imaging patterns are also described on CT and MRI [34]. Messori et al. reported a dermoid that showed signal changes in T1-WI during follow-up changing from hypointensity to hyperintensity related to alterations of the dermoid cyst contents [24]. Intra-operatively, the differentiation between both tumours (dermoid vs. epidermoid) is straightforward once remnants of dermal appendages such as hair and sebaceous glands

are found. In our patients 2, 3 and 4, the pre-operative imaging showed signal characteristics better compatible with epidermoids than with dermoids with T2-WI hyperintensity and T1-WI non-homogeneous hypointensity in the solid tumour parts. The ruptured particles had a homogeneous, fatty aspect on MRI with strong T1-WI hyperintensity (patient 2 and 3) and intraventricular fat/fluid levels on CT (patients 1, 2 and 3). According to the literature, this is not a surprising finding *per se* [45], but the high percentage of patients with atypical MRI signals (71%) in our series justifies critical re-evaluation of the diagnostic radiological criteria.

Patient 1 stands out because the long follow-up period before and after tumour resection is quite unusual. Yaşargil et al. discussed two patients that had a clinical history of 10–20 years before presentation, and Rubin et al. studied one patient with a long history of 15 years before diagnosis and 9 years of follow-up [33, 47]. Epileptic seizures are a common presentation as mentioned previously. Our patient developed left-sided anosmia over the years. To the best of our knowledge, olfactory dysfunction due to the compressive effect of an enlarging dermoid has not been described in the literature. After 10 years of observation, the tumour was radically resected but hyposmia persisted. Nine years after the successful operation, the patient presented with a new onset of seizures. At that point, tumour re-growth was excluded and reactive parenchymal gliosis may have led to his late symptoms. This underlines the fact that despite radical tumour removal, epileptic seizures may recur within some years which has been reported to take place in 5% of all craniotomies for any type of pathology. In any event, complete excision should only be attempted when preservation of neurovascular structures is possible. Dermoids adhesive to critical surrounding structures should be decompressed as far as reasonably achievable, and its capsule should be left behind. If total resection cannot be achieved, gentle and copious irrigation of the tumour cavity was recommended by Ecker et al. in order to prevent delayed vasospastic events [11].

Patient 4 presented with a variety of cerebellar symptoms which were clearly related to the right-sided intraaxial solid dermoid and which resolved after resection of the mass. Infra-tentorial dermoids are known to present in the first two decades of life rather than at 60 years of age [18]. This patient also presented with leptomeningeal enhancement which was interpreted as a diffuse manifestation of aseptic meningitis. Whether or not the meningitis was related to the dermoid remains a matter of debate. However, in the literature various ischaemic events are described in the setting of dermoids and their clinical presentations [11, 14, 26]. In these patients, asymptomatic aseptic meningitis could have initiated reactive vasospasm manifesting as small infarcts in unusual locations.

There is a very large variety of clinical features and imaging aspects of intracranial dermoids. Comparing the findings of previous studies with our results, we would like to emphasise the diagnostic quandaries with MRI and CT [22, 24, 36, 38, 45]. Mixed signals were observed according to most published data. Although very recently some reports have presented data with "non-classical" MRI and CT findings in dermoids, we aim to emphasize the importance of using an MRI protocol involving T2-WI, T1-WI, T1 fat saturated-WI, magnet resonance angiography (MRA) and DWI. Diffusion-weighted imaging facilitates detecting tumour remnants or re-growth after resection. It may also help to identify solid tumour components that cause a decrease in water proton diffusion, resulting in DWI hyperintensity as seen in other keratin-rich lesions. Therefore, this protocol should be used for pre-operative diagnostics and during follow-up to monitor potential tumour re-growth.

A variety of clinical presentations can occur due to rupture of dermoids [1, 12, 14, 16, 17, 20, 23, 24, 26, 30–32, 36–40, 42, 43, 45]. In patient 1, dermoid rupture into the ventricles never led to hydrocephalus. In contrast, ventricle size progressed post-operatively in case 2. Right-sided dilatation of the lateral ventricle was pre-existing, but intra-operative distribution of fatty particles probably caused full-blown occlusive hydrocephalus. The fatty intraventricular components may obstruct the Foraminae of Monro, the Foraminae of Luschka and Magendie or the aqueduct; furthermore, intra-operative retraction is a possible explanation for further displacement of fatty droplets and consecutive hydrocephalus [17, 20, 23]. If feasible, an *en bloc* resection of the dermoid is therefore desirable. The subarachnoid dissemination of fatty particles has probably led to symptoms in patients 2 and 3, with the dermoids being silent until their rupture. Yaşargil et al. have emphasised that communicating hydrocephalus can emerge as a consequence of aseptic meningitis/arachnoiditis [47]. Therefore, close monitoring of ventricle size is essential with this diagnosis.

Conclusion

This retrospective review of 7 histopathologically verified dermoids demonstrates the wide range of clinical presentations. Reactive gliosis can lead to very late post-operative symptoms such as epileptic seizures. Hydrocephalus is a prominent complication, demanding close monitoring of ventricle size. Diffusion-weighted imaging provides essential information about the existence of solid tumour components

pre- and post-operatively. Complete removal of dermoids without dissemination of the contents should be achieved without injury to surrounding neurovascular structures in order to prevent re-growth and associated morbidity.

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Comment

Orakcioglu et al. submit an interesting report on their experience with intracranial dermoid cysts and give an nice overview on the current literature.

As intracranial dermoid cysts account for less than approx. 0.25% of all intracranial neoplasms, they are up 10 times less common than e.g. intracranial epidermoids. That is why a collection of 7 histologically proven cases seems worth publishing to me. The authors summarize all important information on the different clinical signs and symptoms of ruptured dermoid cysts. They conclude, that long-term follow-up is necessary because of the likelihood of post-op hydrocephalus. I completely support the author's opinion, that DWI scans are the most useful imaging modality for diagnosis and follow-up exams.

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